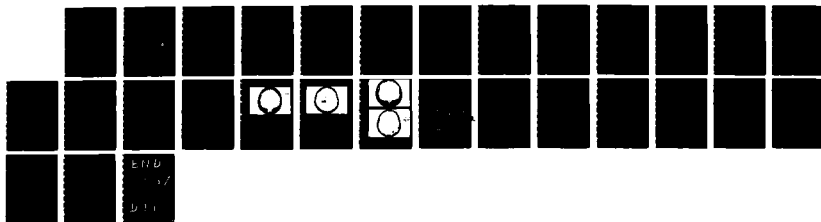


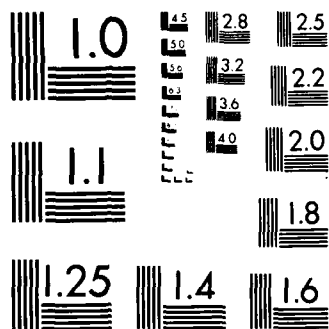
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DEFICITS IN HUMAN VISUAL SPATIAL ATTENTION
FOLLOWING THALAMIC LESIONS

Robert D. Rafal
Roger Williams General Hospital and
Brown University Program in Medicine, Providence

and

Michael I. Posner
McDonnell Center for Studies of Higher Brain Function
Washington University, St. Louis

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Following Thalamic Lesions*

Robert D. Rafalt

Division of Neurology

Roger Williams General Hospital and

Brown University Program In Medicine

Providence, RI 02902

Michael I. Posner

McDonnell Center for Studies of Higher Brain Function and

Departments of Neurology, Neurological Surgery and Psychology

Washington University

St. Louis, MO 63110

(Selective attention / visual attention / thalamic lesions)

A-1

ABSTRACT

There has been recent speculation concerning the role that thalamic nuclei play in directing attention to locations in visual space (Crick, F., 1984 Proc. Nat. Acad. Sci. USA 81, 4586-4590). We measured covert shifts of visual attention in three patients with unilateral thalamic hemorrhages both shortly after the lesion and after a six month delay. The experiment measured reaction time to targets that occurred at locations to which attention had been previously cued (valid trials) or at a currently unattended location (invalid trials). Although the patients showed no deficits in visual fields with perimetry and no neglect in the six month followup, we found slow reaction times for targets on the side contralateral to the lesion whether or not attention had been cued to that location. Deficits have also been found in this task with cortical and midbrain lesions, but the patterns of performance are quite different. The results with thalamic patients suggest they have a specific deficit in the ability to use attention to improve the efficiency of processing visual targets contralateral to the lesion (engage operation). This finding is in accord with ideas of a thalamic link between cortical visual attention and pattern recognition systems (Crick, 1984).

INTRODUCTION

In recent years a number of specific experimental methods have been used with alert monkeys (1-3) and humans (4-6) that time lock covert shifts of attention to the presentation of cues. In neurophysiological studies the orienting of attention is inferred from selective enhancement in neuron firing rate in response to the cue. Cognitive studies measure the allocation of attention in terms of improved efficiency in responding to signals at the cued locations in comparison to other spatial locations. These approaches have begun to converge to identify the neural mechanisms controlling visual attention. Cognitive studies with normal humans using visual cues to direct attention covertly to a location eccentric from fixation show more efficient processing of signals at the cued location. This enhancement includes lowered manual (5) and saccadic (7) reaction times, reduced sensory thresholds (8), improvement in conjoining features (9) and modulation of evoked electrical potentials recorded from the scalp (10). These observations support the concept of attention as a mechanism for relative enhancement of information processing at a selected spatial location. There is also evidence that the area of enhancement becomes larger as cues are presented more eccentrically in correspondence with the known characteristics of the neural magnification factor (11,12).

Areas of the monkey brain showing selective neuronal enhancement include the posterior parietal lobe (1,2), the superior colliculus (2) and substantia nigra (pr) (13) of the midbrain, and the lateral pulvinar (14). The same visual cueing method described above was used to demonstrate that modulation of GABAergic transmission in thalamus (with iontophoretic injections of muscimol or bicuculline) systematically affect the orienting of attention contralaterally (15). Reaction time studies using cueing in neurologic patients have confirmed that lesions of the parietal lobe (16) and peritectal regions of the midbrain (17) produce distinctly different deficits in orienting visual attention.

Three computations have been suggested in orienting of visual attention. First attention must "disengage" from the current location; then "move" to a new location; then "engage" at the new location. Deficits in each of these three elementary operations can be identified in cueing studies. At the beginning of the trial the subject is maintaining fixation at the center of the display without actively attending to any spatial position (no targets occur at the center). When the cue is presented the subject must move attention to the cued location and engage attention there in anticipation of the forthcoming

target. The efficiency of moving attention can be inferred, then, from the rate of improvement of RT with cue-to-target delay on valid trials. A deficit in the move operation can be inferred by a deficiency (i.e. a delay or reduction) in this improvement.

A deficit in the move operation has been found in patients with progressive supranuclear palsy who have degeneration of the superior colliculus and peritectal region (19,20). In these patients saccadic eye movements are relatively more impaired in the vertical dimension than are horizontal eye movements. We therefore compared vertical and horizontal attention shifts. RT on valid trials improved more slowly with time following the cue in the vertical dimension.

A different pattern of results was shown for patients with parietal lesions (16). Reaction times improved at the same rate in both visual fields following a valid cue. This indicates that parietal lesions do not slow the movement of attention toward the contralateral field. Moreover, the asymptote of these functions differed very little between fields showing that the ability to use attention to engage the target location did not differ greatly between visual fields. In contrast to the midbrain patients there was a dramatic increase in RTs to targets in the contralateral field following invalid cues. According to our scheme, if attention is shifted to the cue but the target appears elsewhere, it is necessary to disengage attention from the cue before moving to the target. The selective slowing of detection RT in the invalid cue condition suggests, therefore, that the parietal lobe plays a special role in mediating the disengage operation.

Parietal lesions and midbrain lesions have distinctly different effects on orienting attention: midbrain lesions appear to produce a specific deficit in the move operation; whereas parietal lesions selectively appear to produce a specific deficit in the disengage operation.

We now extend the use of cueing paradigms to measure attention shifts in three neurological patients with thalamic hemorrhages. This method permits us to compare the thalamic deficit with those found in midbrain and parietal patients. Lesions of any of these areas can produce clinical symptoms of neglect of contralateral stimuli (19). However, the computations performed by these areas may be quite different. If the patterns of performance deficit due to lesions of these areas differ, it should be possible to further the analysis of the role of each area.

METHODS

Subjects

Three patients with hemorrhages in the thalamus were studied in an experiment to measure covert shifts of visual attention on two occasions: in the acute stage they were tested as soon as they were able to perform the task; each was retested after 4-6 months of recovery (chronic stage). Patient VM, a 65 year old man, had a large hemorrhage centered in the left thalamus with rupture of the hemorrhage into the ventricular system. He was initially comatose with right hemiplegia, hemianesthesia and ocular deviation. Fig. 1 shows the CT scan findings at the time of initial testing seven weeks after the ictus. At that time he still manifested some psychomotor retardation and mild visual neglect. At the time of retesting six months after the ictus, he was alert, lucid and subtle visual neglect was evident only on a letter cancellation task. The other two patients had smaller lesions which did not impair alertness, and were first tested in the second week of their illness. Patient VL, a 67 year old woman, had a hematoma in the right thalamus (Fig. 2). Patient NA, a 54 year old man, had a small hematoma in the right thalamus involving the nuclei centromedianum, ventrolateral and lateral posterior (Fig. 3A). The hemorrhage extended into the posterior limb of the internal capsule, and ventral to the thalamus into the region of the zona incerta and perigeniculate region (Fig. 3B). Patients VA and NL had hemiparesis and hemisensory impairment contralateral to their lesions. Neither had any signs of visual neglect on detailed clinical testing. At the time of follow up testing 4-6 months after their strokes, perimetry testing confirmed that the visual fields were intact in all three patients.

Procedure

Subjects sat facing a video display screen with one finger of the preferred hand on a response key placed on a table between the subject and the display. Light pressure on the key activated a microswitch which recorded RT. The display consisted of a + sign at the center, flanked five degrees to left and right by one-degree unfilled squares. Subjects were instructed to maintain gaze on a plus sign in the middle of the screen and not to move the eyes. Eye position was monitored with a closed circuit video camera to assure that the eyes remained fixed at the center. Subjects practiced the task before data was collected while the experimenter observed to ascertain that the directions were understood and the subject was not moving the eyes. The intertrial interval was 2 seconds. At the start of each trial the fixation point was extinguished and .5 second later the cue was

presented by brightening, randomly and with equal probability, one of the two peripheral boxes. The cue remained visible for 300 msec. After an interval (50, 150, 500 or 1,000 msec) following the onset of the cue, a target appeared either at the cued location or in the opposite visual field. Subjects were instructed to press the response key as quickly as possible any time the target (a bright asterisk filling one of the peripheral boxes) appeared. The target remained visible until the subject responded (or for 5,000 msec). In this experiment, the target was on the cued side on 80% of trials (valid trials), while on 20% of trials, the target appeared in the box contralateral to the cue. The probabilities were designed to induce the shift and maintenance of attention to the cued location. Since the eyes remained fixed at the center, and since the motor response (a simple key press) was always the same, any difference of RT between valid and invalid cue conditions may be assumed to index a covert movement of attention to the cued location.

RESULTS

We first excluded all RTs less than 100 or greater than 4,000 millisecc. Only a very few times were affected by this rule. The median RT for each patient in each condition was calculated.

A within factor analysis of variance was run with the following factors: stage of illness (acute vs. six month followup); target field (contralateral to lesion vs. ipsilateral to lesion); cue validity (target appeared at cued location (valid) versus at uncued location (invalid); and cue to target interval (50, 150, 550 or 1000 msec.).

When tested in the chronic stage (six months or more after the lesion) the patients were faster than in the acute stage but this did not reach statistical significance ($F[1,2] = 2.65$). Thus we display the combined data for acute and chronic tests in Fig. 4.

Reaction times are faster in the ipsilateral field than in the contralateral field for both validity conditions ($F[1,2] = 36.8$, $p < .025$). Validity has a significant effect with valid targets (solid lines) responded to faster than invalid targets (dashed lines) $F[1,2] = 23$, $p < .05$) and validity interacts with interval such that its effects are greater at short cue to target intervals ($F[3,6] = 8$, $p < .025$). Finally, this interaction of validity and interval is significantly greater in the contralateral visual field than in the ipsilateral field resulting in a triple order interaction between validity, field and interval ($F[3,6] = 11$, $p < .01$).

These results would be consistent with a primary visual defect in our patients. However, our thalamic patients had no clinical evidence of visual impairment and, as mentioned, clinical neglect was not conspicuous (and was totally absent in two of the patients). All three patients showed no contralateral visual field defect on formal perimetric examination, even with the smallest (3mm) target. Since the target in our experiment was a large (1 degree), bright signal presented in the parafoveal (5 degree eccentricity) region, it seems very unlikely that a subtle visual field defect, beyond the sensitivity of perimetric testing, could have accounted for the dramatic slowing of contralesional detection RT. A fourth patient with a posterior cerebral artery stroke syndrome and CT evidence of infarction in the right thalamus and occipital lobe was also tested. He had a dense homonymous hemianopia, and could not respond to any signal presented in this contralesional visual field. He was tested in an experiment where all cues and targets were presented in his intact visual field ipsilateral to the lesion (20). The target was presented at the same location on each trial, but was preceded by a cue which first summoned attention either to the left or right of the forthcoming target. On each trial, then, he had to disengage his attention to move it in either an ipsilesional or contralesional direction. When he had to shift attention leftward (contralesionally) detection RTs were systematically longer than when he had to shift attention rightward (ipsilesionally). This result, obtained entirely within the intact visual field, could not have been due to differences in visual sensitivity since the target always occurred at the same location.

DISCUSSION

There are three salient features of the data depicted in Fig. 4. 1) For the valid trials, the cue produces a similar improvement in RT, as a function of cue-target interval, in both visual fields. 2) For the invalid trials, there are slow RTs in the contralesional field for the short cue-target intervals. 3) There is a dramatic main effect of visual field, with mean RT to contralesional targets being substantially slower. Consideration of these three findings in comparison to previous findings for patients with midbrain and parietal lobe lesions, provides insights into the role of the thalamus in a distributed neural system for orienting visual attention.

Inspection of the data from the valid cue condition (solid lines) reveals a decrease in RT with interval. Although RT is slower for all contralesional targets, the improvement in RT from valid cues over time is equivalent in the two hemifields. This pattern for valid cue trials differs from what we have found previously in patients with midbrain

lesions in whom we have argued for a disorder in the "move" operation. In midbrain patients the improvement of RT on valid trials was slower in the affected direction (vertical). Thus, midbrain patients were slow in moving attention. In contrast, for the thalamic subjects RT to valid trials improves following the cue with a similar time course in both visual fields. In contrast to the midbrain lesion patients, they do not appear to have a deficit in moving attention in response to cues.

The second feature of these results are the long RTs on the invalid trials relative to valid trials in the contralesional field for the short cue-target intervals. This pattern is similar to that found in our parietal patients, and suggests that thalamic lesions do affect the disengage operation in a qualitatively similar way. Indeed, the mean reaction time to invalidly cued contralesional targets for these early cue-target intervals in the thalamic lesion patients is similar to that previously for patients with right parietal lesions. Nevertheless, the relative slowing on invalid trials when compared to validly cued targets in the same field is much less in the thalamic patients. Moreover, the disengage deficit in the parietal lesion patients persisted even through the longest (1000 msec) cue-target interval. In the thalamic lesion patients, the disengage deficit is manifest only at the early cue-target intervals, while the cue is still present. We conclude that, although intact thalamic function may be necessary for disengaging attention, the parietal lobe is chiefly responsible for this operation. The thalamic lesion may have an indirect affect on parietal function to produce the "disengage" deficit.

In spite of their apparent ability to move their attention in response to the cue, the third and most striking aspect of the data is the persisting main effect of visual field for both valid and invalid targets. Even at the 1000 msec cue-target interval, when attention has had time to reach the target location, RT to detect contralesional targets remains slower, and at no time is this difference less than 200 msec. This difference between the two visual fields is about four times as long as the mean difference which we found for parietal lesion patients (16). Only one of those thirteen parietal patients showed a RT for validly cued contralateral targets at the 1000 msec cue-target interval which was as long as the mean for the three thalamic patients. The different pattern of results for the valid cue condition for the thalamic lesion patients, in comparison with that seen with midbrain or parietal lesions, is consistent with a deficit in the engage operation.

The different pattern of experimental results between parietal and thalamic lesion patients is especially interesting when one considers that, even though the thalamic patients had much slower contralesional detection RTs than parietal patients in the valid cue condition, most of the parietal lesion patients had more clinical neglect than did any of the thalamic lesion subjects. The fact that these patients show less clinical neglect than do parietal lesion patients, whose deficit lies in the disengage operation, leads us to speculate that clinical neglect, an important source of disability, can be linked most directly to a disorder in the disengage operation.

It is not possible to make precise inferences about the specific neural structure responsible for the effects found in our patients. In two patients the hemorrhage involved large parts of the thalamus, including the pulvinar, as well as adjacent structures. The most restricted lesion was present in patient NA, who also had the least severe clinical impairment. Since this patient had the same pattern of results as the thalamic group as a whole, both at acute and chronic testing, the anatomic localization of his lesion on CT (Fig. 3) provides the best information bearing on this question. The lesion involves the nuclei lateral posterior, centromedianum and ventrolateral. Unlike the other two patients, it does not clearly involve the pulvinar (Fig. 3A). It extends ventral to the thalamus and involves the perigeniculate region (Fig. 3B).

This area may correspond to the region of the perigeniculate nucleus considered by Crick as possibly mediating the "searchlight" of visual attention. This structure, related to the thalamic reticular nuclei, sends GABAergic projections to the dorsal thalamic nuclei which may gate their processing of sensory information. Petersen et al (15), using the experimental task described here in monkeys, have shown that manipulation of GABAergic transmission to pulvinar, with iontophoretic injections of muscimol or bicuculline, systematically affect the orienting of attention contralaterally. It would be of interest to compare, in experimental animals, the effects of discrete lesions of pulvinar and of the thalamic reticular region, in this task.

Recent PET scan studies in patients with thalamic lesions show that these lesions produce diffuse hypometabolism throughout the ipsilateral hemisphere (21). These results suggest that the thalamus is involved in cortical "activation" in some way. Whether such activation can be interpreted in terms of a defect in attention, in the sense applied in this communication, remains conjectural. The hypometabolism (21) was most pronounced in the acute phase, and had diminished substantially within 4-6 months.

According to current neurobiological views, the visual cortex involves somewhat separate areas for signal localization and directing of visual attention (parietal) than for pattern recognition (occipito-temporal) (22). We have shown that patients with parietal lesions have defects in pattern recognition on the side contralateral to the lesion (23). This suggests that the ability to recognize patterns rests in part upon an intact visual attention system. The route by which the parietal system interacts with the pattern recognition system is not known. The current results agree with the ideas of others that thalamic nuclei may play a role in this interaction (3,24). Moreover, it suggests that the thalamic effects on attention are not due to remote effects on cortical or midbrain areas alone. Our evidence is that thalamic lesions produce a different pattern of deficit than found for midbrain or cortical lesions. Thus the computations performed by thalamic structures are distinct and do not appear to be an indirect reflection of damage elsewhere. Even closer contact between human studies and alert monkey studies should be useful in developing a more complete model of how these neural systems interact in orchestrating a shift of visual attention.

FIGURE LEGENDS

FIG 1: CT scan from patient VM at the time of acute phase testing seven weeks after his stroke. There is a resolving large hematoma (arrow) centered in the left pulvinar.

FIG 2: CT scan from patient VL at the time of her stroke showing a large hematoma in the posterior right thalamus.

FIG. 3: CT scan from patient NA at the time of his stroke. A. There is a small hematoma in the right thalamus centered in the ventrolateral nucleus and involving nuclei lateral posterior and centromedianum. B. The hematoma extends into the posterior limb of the internal capsule, and ventral to the thalamus into the area of the zona incerta and into the perigeniculate region (arrow).

FIG. 4: Mean RT for three thalamic patients as a function of cue-target interval. Stimulus onset asynchrony between cue and target in millisec.

FOOTNOTES

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† Reprint requests should be addressed to:

Robert D. Rafal
Roger Williams Hospital
Department of Neurology
825 Chalkstone Avenue
Providence, RI 02902

‡ Localization of the lesions was determined by relating the CT findings to De Armond SJ, Fusco MM and Dewey MM: Structure of the Human Brain: A Photographic Atlas (Second Edition) New York: Oxford University Press, 1976.

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FIG 1

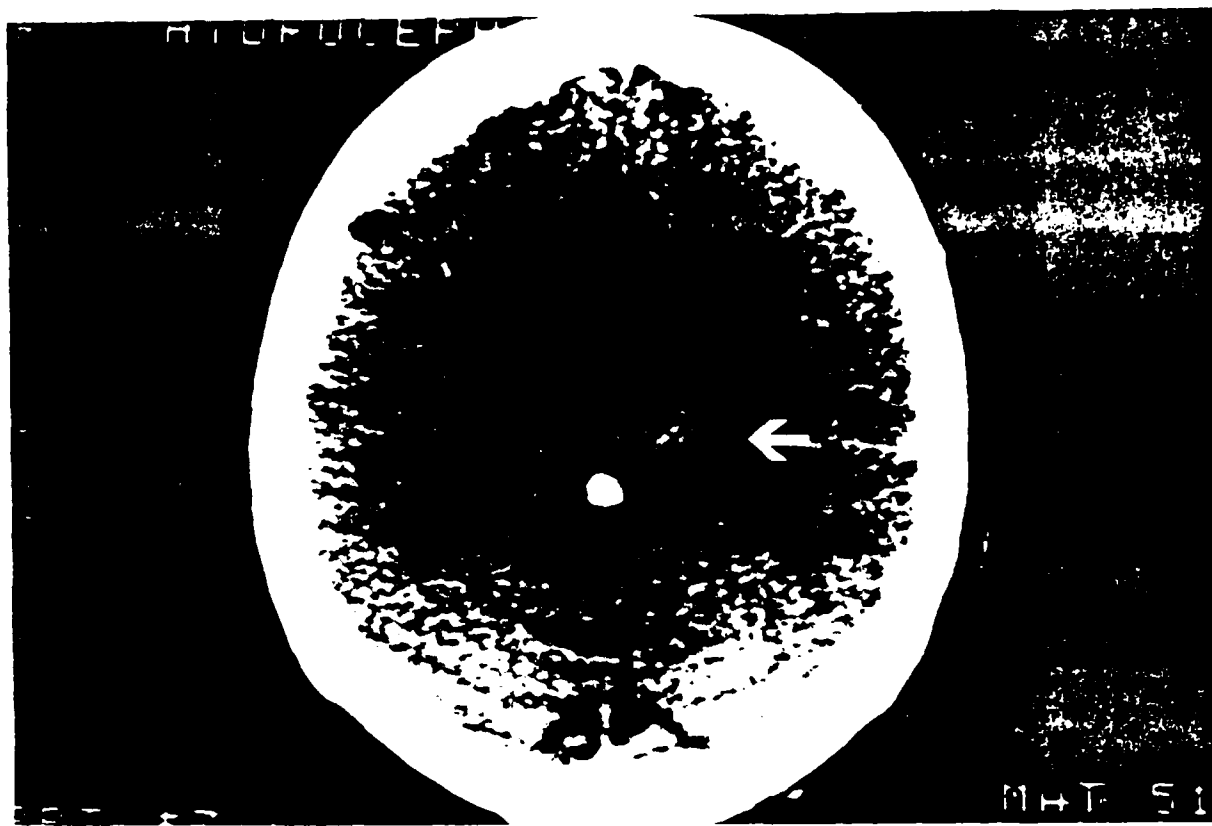


FIG 2

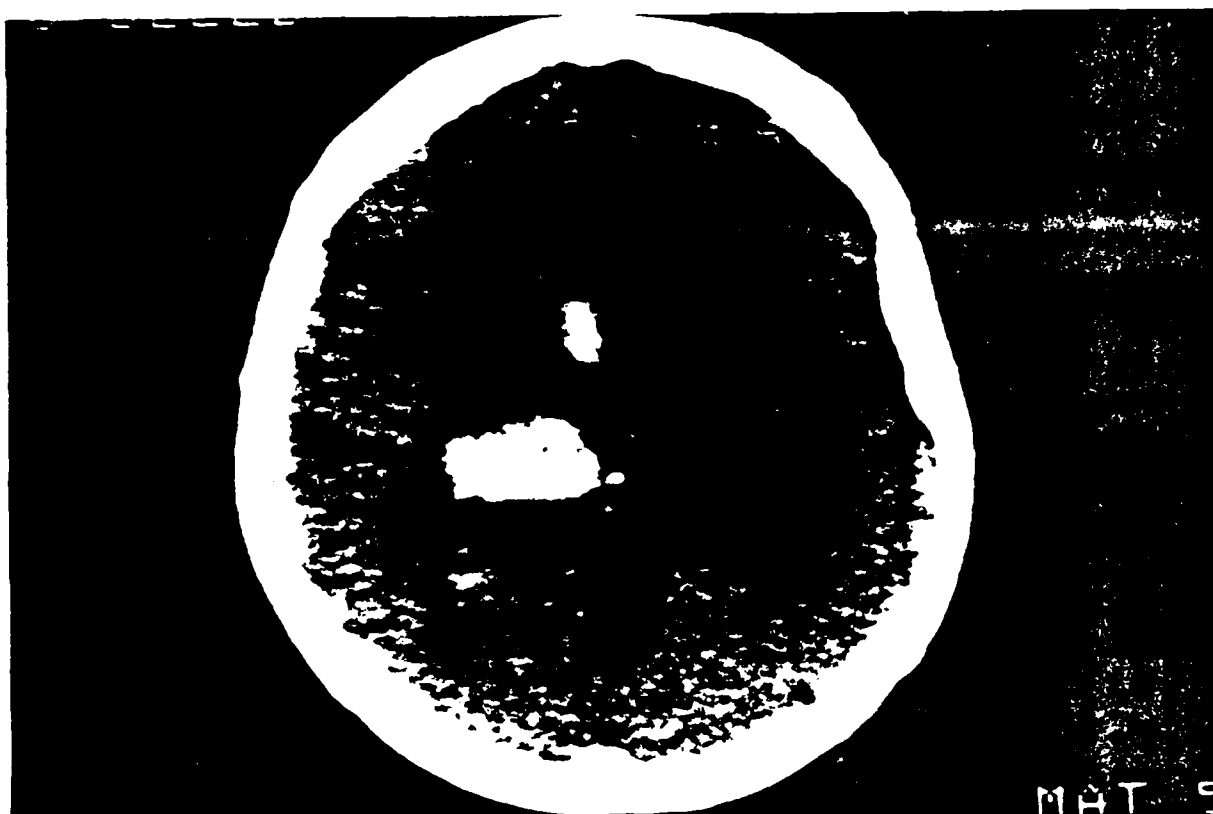


FIG 3A

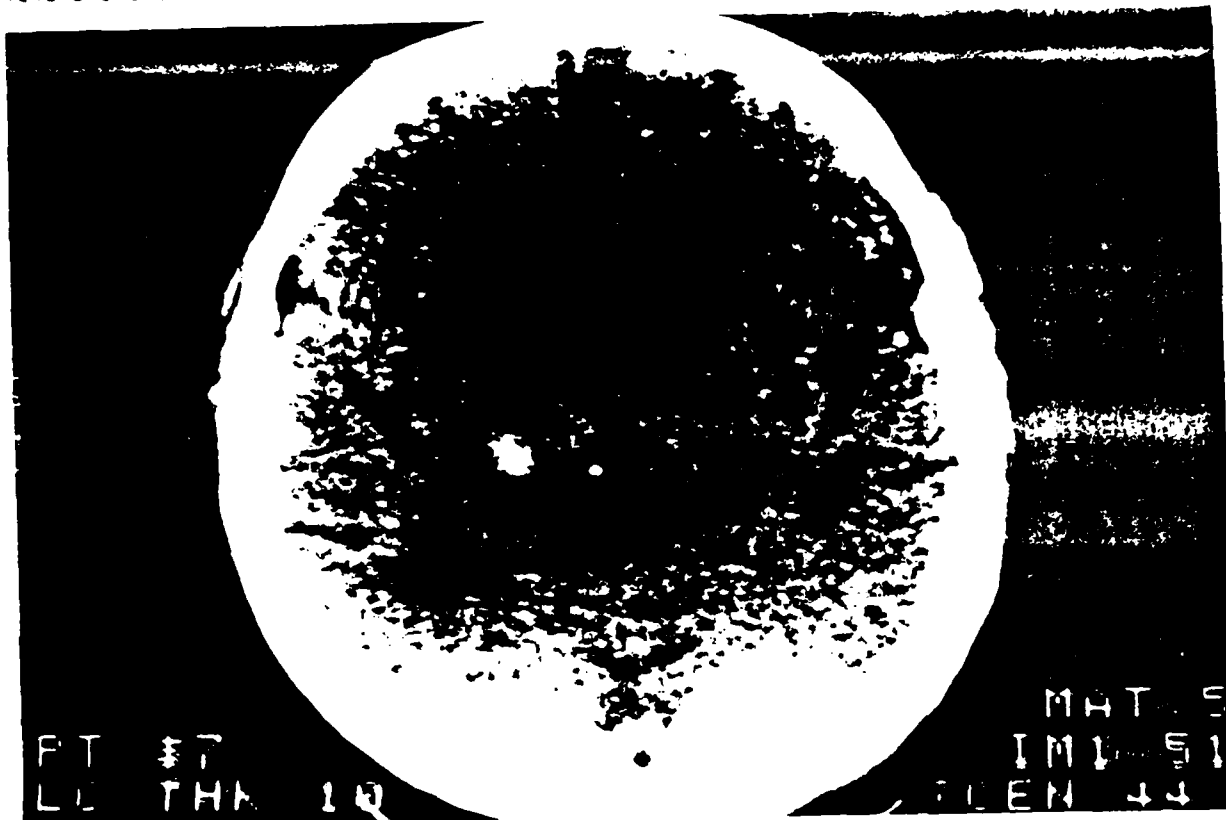


FIG 3B

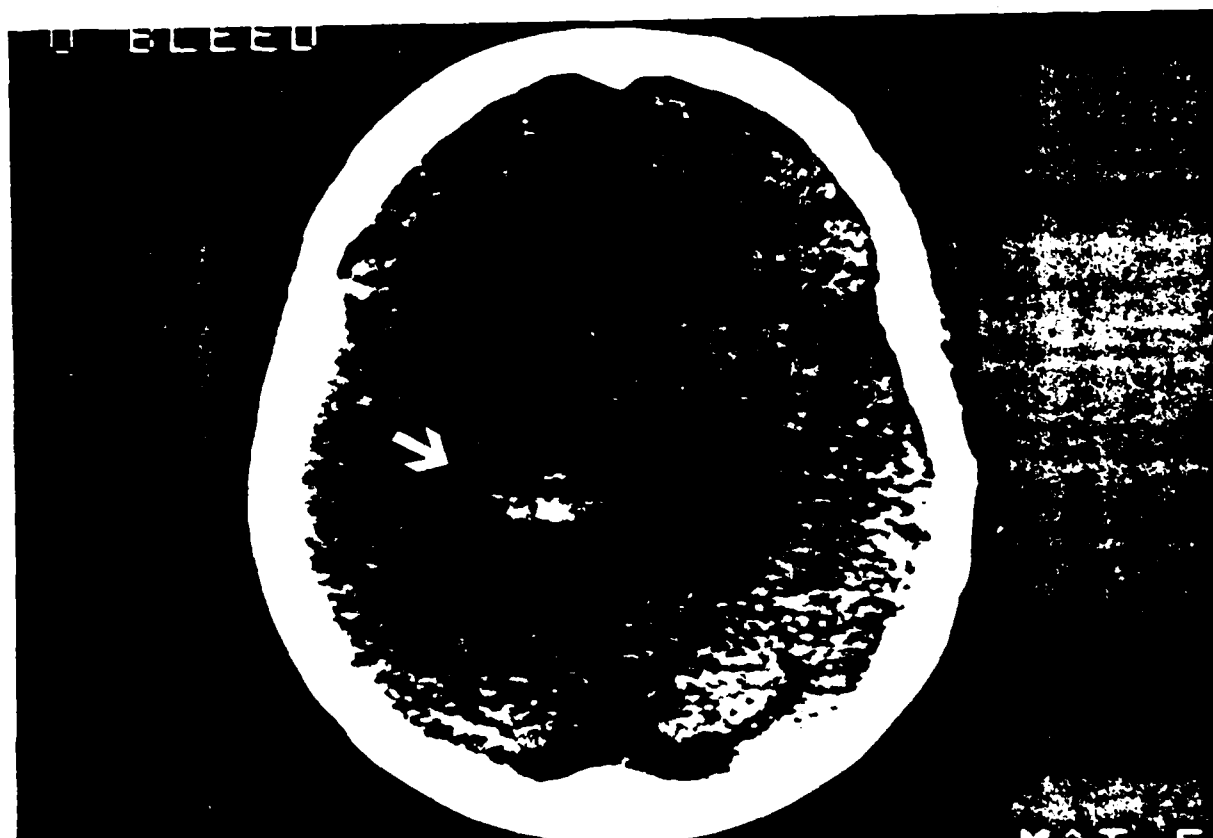
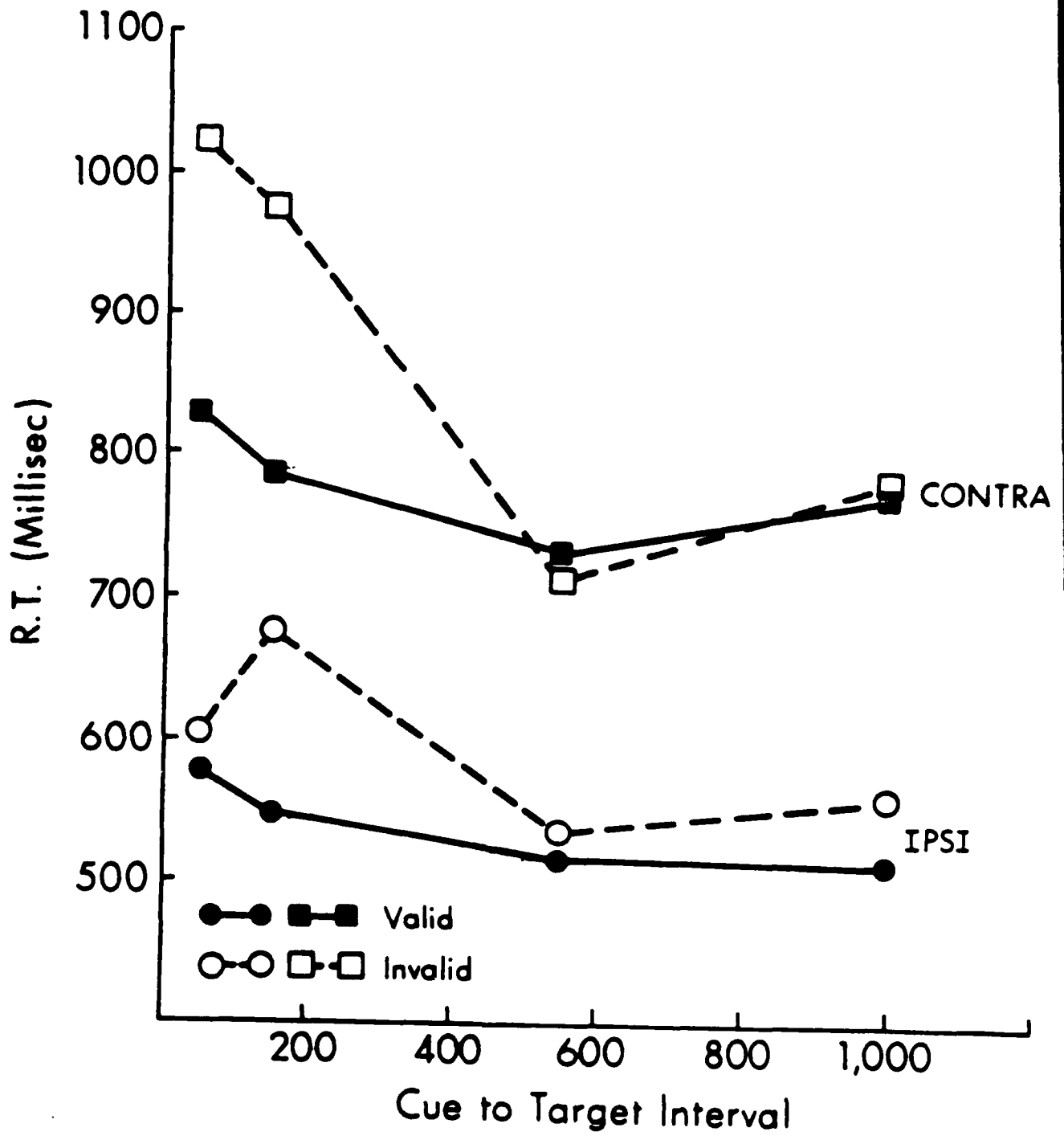


Figure 4



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360 Huntington Avenue
Boston, MA 02115

Dr. Pat Carpenter
Carnegie-Mellon University
Department of Psychology
Pittsburgh, PA 15213

Dr. Tyrone Casman
American Society of
Cybernetics
3428 Fremont Ave. South
Minneapolis MN 55408

Dr. Alphonsie Chapanis
8415 Bellona Lane
Suite 210
Baltown Towers
Baltimore, MD 21204

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OUSDME
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800 M. Quincy Street
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Center for Neural Science
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Learning R&D Center
University of Pittsburgh
3939 O'Hara Street
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Naval Undersea Warfare Engineering
Keyport, WA 98345

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Committee on Human Factors
National Academy of Sciences
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505 Haddon Road
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Champaign, IL 61820

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Suite 400
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75 E. River Road
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Department of Psychology
Community College of
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2 Washington Square Village
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Training Research Division
NMBRO
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Behavioral Technology
Laboratories - USC
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Redondo Beach, CA 90277

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University of Pittsburgh
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Army Research Institute
5001 Eisenhower Avenue
Alexandria, VA 22333

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Institute for Defense Analyses
1801 M. Beauregard St.
Alexandria, VA 22311

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Department of Psychology
E10-018
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Department of Psychology
Campus Box 346
University of Colorado
Boulder, CO 80309

Dr. Peter Polson
University of Colorado
Department of Psychology
Boulder, CO 80309

Dr. Steven E. Poltrook
MCC
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Medical School
St. Louis, MO 63110

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Department of Psychology
MIT (E-10-032)
Cambridge, MA 02139

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Stanford University
Department of Psychology
Bldg. 4201 -- Jordan Hall
Stanford, CA 94305

Dr. Joseph Psotka
ATTN: PENI-1C
Army Research Institute
5001 Eisenhower Ave.
Alexandria, VA 22333

Dr. Mark D. Reckase
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Carnegie-Mellon University
Schenley Park
Pittsburgh, PA 15213

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University of Maryland
School of Medicine
Department of Neurology
22 South Greene Street
Baltimore, MD 21201

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New School for Social Research
65 Fifth Avenue
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Alexandria, VA 22333

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Department of Psychology
Yale University
Box 11A, Yale Station
New Haven, CT 06520

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Department of Psychology
3815 Walnut Street
Philadelphia, PA 19104

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Bolt Beranek & Newman, Inc.
10 Moulton St.
Cambridge, MA 02238

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Training Research Division
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1100 S. Washington
Alexandria, VA 22314

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Perceptronics, Inc
6271 Varrel Avenue
Woodland Hills, CA 91364

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Yale University
Department of Psychology
Box 11A, Yale Station
New Haven, CT 06520

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Yale University
Computer Science Department
P.O. Box 2158
New Haven, CT 06520

Dr. Walter Schneider
Learning R&D Center
University of Pittsburgh
3939 O'Hara Street
Pittsburgh, PA 15260

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Institut fuer Psychologie
der RWTH Aachen
Jaegerstrasse zwischen 17 u. 19
5100 Aachen
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Stanford University
Department of Psychology
Bldg. 4201 -- Jordan Hall
Stanford, CA 94305

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Fairfax, VA 22030

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Washington, DC 20380

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Assessment Systems Corp.
2233 University Avenue
Suite 310
St. Paul, MN 55114

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Department of Psychology
Carnegie-Mellon University
Schenley Park
Pittsburgh, PA 15213

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Navy Personnel R&D Center
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Division of Psychological Studies
Educational Testing Service
Princeton, NJ 08541

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Boit Beranek & Newman, Inc.
50 Houlton Street
Cambridge, MA 02138

Dr. Norman M. Weinberger
University of California
Center for the Neurobiology
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Irvine, CA 92717

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M660 Elliott Hall
University of Minnesota
75 E. River Road
Minneapolis, MN 55455

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1325 J. R. Lynch Street
Jackson, MS 39217

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Chapel Hill, NC 27514

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Code 6021
Warminster, PA 18974-5000

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U.S. Army Institute for the
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5001 Eisenhower Avenue
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1275 York Avenue
New York, NY 10021

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Office of Naval Research
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Arlington, VA 22217-5000

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Navy Personnel R&D Center
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Captain P. Michael Curran
Office of Naval Research
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Code 125
Arlington, VA 22217-5000

Defense Technical Information Ctr.
Cameron Station, Bldg. 5
Alexandria, VA 22314
Attn: TC

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Bolt Beranek & Newman
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Institute for Cognitive Science
University of California
La Jolla, CA 92093

Dr. Steven Zornetzer
Office of Naval Research
Code 1140
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Arlington, VA 22217-5000

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Department of Psychology
Pittsburgh, PA 15213

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Orlando
NPRDC Liaison Officer
NTSC Orlando, FL 32813

Dr. Joel Davis
Office of Naval Research
800 North Quincy Street
Code 1141NP
22217-5000

ERIC Facility Acquisitions
4833 Rugby Avenue
Bethesda, MD 20014

J. D. Fletcher
9931 Corsica Street
Vienna, VA 22180

Dr. David Navon
Institute for Cognitive Science
University of California
La Jolla, CA 92093

Dr. Robert Sasmor
Army Research Institute
5001 Eisenhower Avenue
Alexandria, VA 22333

Dr. Michael J. Zyda
Naval Postgraduate School
Code 52CK
Monterey, CA 93943-5100

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